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Invited Review

# Parasites and the conservation of small populations: The case of *Baylisascaris procyonis*

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ABSTRACT

Human demands on natural resources result in landscape changes that facilitate the emergence of disease. Most emerging diseases are zoonotic, and some of these pathogens play a role in the decline of vulnerable wildlife species. *Baylisascaris procyonis*, the common roundworm parasite of raccoons (*Procyon lotor*), is a well recognized zoonotic infection that has many of the properties associated with a pathogen capable of driving extinction. It is highly non-specific and frequently pathogenic with regard to paratenic hosts, which contact eggs of *B. procyonis* at raccoon latrines. Eggs accumulate at latrines and remain viable for many years. Transmission of *B. procyonis* is sensitive to changes in land-use, and fragmented habitats increase contact rates between raccoons, potential paratenic hosts, and the parasite. Raccoons, and subsequently *B. procyonis*, have been introduced to Europe and Japan, where naïve vertebrates may be exposed to the parasite. Finally, domestic animals and exotic pets can carry patent infections with *B. procyonis*, thus increasing environmental contamination beyond raccoon latrines, and expanding the area of risk to potential paratenic hosts. This parasite can potentially contribute to extinctions of vulnerable species, as exemplified by the case of the Allegheny woodrat (*Neotoma magister*), a species that has experienced local declines and extinctions that are linked to *B. procyonis*. Conservation strategies for vulnerable species should consider the transmission ecology of parasitic pathogens, like *B. procyonis*.



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## 1. Introduction

The current human population size and subsequent demands on natural resources have resulted in shifts in land-use patterns and ecosystem processes that have significant implications for the emergence of disease (Daszak et al., 2001; Plowright et al., 2008; Smith et al., 2009). Links between human and environmental health are well documented, and the emergence of numerous

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zoonotic diseases, which now account for approximately 58% of all human pathogens, have been related to changing land use and resource consumption patterns (Woolhouse and Gowtage-Sequeria, 2005). Since the majority of emerging diseases originate in wildlife populations (~72%, Jones et al., 2008), it is important to determine ecological facilitators of disease transmission among wildlife, and the implications of these emerging diseases on wildlife populations themselves. Infectious diseases are important to consider with regard to conservation efforts, and they may play a role in wildlife extinctions (Smith et al., 2009). While only 3.7% of recorded extinctions are directly linked to disease (Smith et al., 2006), some pathogens have particular characteristics that make them more likely to facilitate extinction (Woodroffe, 1999; Daszak et al., 2000; Altizer et al., 2003; Bradley and Altizer, 2007; Pedersen et al., 2007; Smith et al., 2009; Thompson et al., 2010). Pathogens may play a role in facilitating extinctions when one or more of the following conditions occur: (1) the pathogen is relatively non-specific with regard to host (Pedersen et al., 2007), (2) the pathogen remains viable in the environment or reservoir hosts (Smith et al., 2009; Thompson et al., 2010), (3) habitat loss or overexploitation results in small or fragmented host populations and/or increased contact rates with pathogens (Woodroffe, 1999; Altizer et al., 2003; Bradley and Altizer, 2007; Smith et al., 2009; Thompson et al., 2010), (4) hosts are artificially dispersed via translocation (Daszak et al., 2000; Smith et al., 2009; Thompson et al., 2010), and (5) domestic animals are involved in transmission dynamics of the pathogen (Smith et al., 2009). The majority of pathogens that meet these criteria are zoonotic microparasites including viruses, bacteria, and protists (Taylor et al., 2001; Pedersen et al., 2007); however, there is increasing evidence that multi-host macroparasites contribute to population declines of some vulnerable host species (LoGiudice, 2003; Polley, 2005; Brearley et al., 2013). One such macroparasite that fits the criteria to facilitate extinctions is *Baylisascaris procyonis*, a common nematode parasite of raccoons (*Procyon lotor*) that is a well-recognized zoonotic infection (Murray and Kazacos, 2004; Gavin et al., 2005; Kazacos et al., 2013) and the most common cause of clinical larva migrans among wild and domestic animal species (Kazacos, 2001; Gavin et al., 2005). Several dozen fatal or severe cases of human baylisascariasis (Huff et al., 1984; Fox et al., 1985; Cunningham et al., 1994; Park et al., 2000; Rowley et al., 2000; Moertel et al., 2001; Gavin et al., 2002; Kazacos et al., 2002; Murray and Kazacos, 2004; Wise et al., 2005; Pai et al., 2007; Chun et al., 2009; Hajek et al., 2009; Mehta et al., 2010; Perlman et al., 2010; Haider et al., 2012; Hung et al., 2012; Kelly et al., 2012; Peters et al., 2012; Kazacos, personal communication 2013) have made this parasite a priority for public health and wildlife management officials (Sorvillo et al., 2002; Roussere et al., 2003; Murray and Kazacos, 2004; Gavin et al., 2005; Page et al., 2009a, 2011; Kazacos et al., 2013). In response to the seriousness of human cases and increasing raccoon densities in close proximity to humans, much of the work on *B. procyonis* has focused on measurements of prevalence (Jacobson et al., 1982; Kazacos and Boyce, 1989; Miyashita, 1993; Kazacos, 2001; Evans, 2002a; Roussere et al., 2003; Page et al., 2005, 2009b; Sexsmith et al., 2009; Blizzard et al., 2010a,b; Kresta et al., 2010; Chavez et al., 2012; Samson et al., 2012; Hernandez et al., 2013), transmission dynamics (Page et al., 1998, 2008, 2009a, 2001a,b; LoGiudice, 2001; Gompper and Wright, 2005; Kellner et al., 2012) and possible strategies to mitigate risk (Page et al., 2011). Fewer studies have examined the implications of *B. procyonis* with regard to conservation and biodiversity (Evans, 2002a), but the parasite is implicated in population declines and local extirpations of Allegheny woodrats (*Neotoma magister*) (McGowan, 1993; LoGiudice, 2006) and possibly other species. This review will focus on the ecology and transmission dynamics of *B. procyonis* and the implications for conservation of wildlife populations.

## 2. *Baylisascaris procyonis* life cycle

*Baylisascaris procyonis* is an intestinal nematode of raccoons, the definitive host (Kazacos, 2001). Infections are common among raccoons and prevalence of infection can be as high as 82% (Kazacos, 2001). Juveniles are susceptible to direct infection via ingestion of eggs; therefore, prevalence can reach >90% among young raccoons (Kazacos and Boyce, 1989). Prevalence of infection among adult raccoons is lower (37–55%) because they become infected primarily via ingestion of paratenic hosts (Kazacos, 2001). Seasonal changes in prevalence have been noted in some regions (Evans, 2002b; Page et al., 2005; Page et al., 2009b) with marked declines during late winter months suggesting a self-cure (Kazacos, 2001). New infections occur in the spring resulting in highest measurements of prevalence in the fall (Kazacos, 2001; Evans, 2002b). Mature female worms produce, on average, over 100,000 eggs/day resulting in an infected raccoon shedding as many as 20,000–26,000 eggs/g feces (Kazacos, 1982, 2001; Snyder and Fitzgerald, 1985). Under optimum conditions, eggs embryonate to the infective stage within 11–14 days, and can remain viable in the environment for years (Kazacos and Boyce, 1989; Kazacos, 2001). When small mammals or birds ingest eggs, the larvae emerge and begin an aggressive somatic migration (Kazacos, 2001), with a low percentage (5%) entering the central nervous system (CNS) causing damage, clinical disease and death (Tiner, 1953; Sheppard and Kazacos, 1997). Larvae within the CNS or encapsulated in visceral or somatic tissues of paratenic hosts will infect raccoons when the tissue is consumed (Kazacos, 2001).

## 3. Non-specificity of *B. procyonis* larval infections

*Baylisascaris procyonis* is highly non-specific with regard to paratenic hosts and over 130 species of vertebrates have been identified with clinical larval infections (Kazacos, 2001; Kazacos, personal communication 2013). Natural infections have been reported across taxa including infections of mammals including rodents, lagomorphs, carnivores, and primates; and birds including galliformes, columbiformes, passeriformes, and psittaciformes (Kazacos, 2001; Evans, 2002a). Captive animals are vulnerable to infection especially when exposed to cages or bedding contaminated with raccoon feces (Sato et al., 2003; Wolf et al., 2007) or when raccoons have access to outdoor exhibits or captive habitats (Ball et al., 1998; Hanley et al., 2006; Gozalo et al., 2008; Thompson et al., 2008). Such outbreaks have affected a range of species including domestic rabbits (Kazacos et al., 1983; Sato et al., 2003), captive birds (Richardson et al., 1980; Kazacos et al., 1982, 1986; Wolf et al., 2007; Thompson et al., 2008), captive rodents (Van Andel et al., 1995; Thompson et al., 2008), and captive primates (Ball et al., 1998; Hanley et al., 2006; Gozalo et al., 2008).

Parasites do not necessarily threaten endangered species when the parasite is a specialist pathogen (Woolhouse et al., 2001); however, generalist parasites that do not have a high level of host-specificity, like *B. procyonis*, could threaten vulnerable populations of (paratenic) hosts (Pedersen et al., 2007). Tiner (1953) demonstrated that larval *B. procyonis* infections resulted in CNS involvement that contributed to 5% mortality of white-footed mice (*Peromyscus leucopus*) populations. While studies of *B. procyonis* induced mortality among wild populations of potential paratenic hosts are limited, all of the >130 noted species of larval infection with *B. procyonis* resulted in CNS involvement usually with severe clinical disease or death (Kazacos, 2001, Kazacos, personal communication 2013), and thus these species could be vulnerable to population declines if coexisting with high densities of raccoons.

## 4. Environmental resilience of *B. procyonis*

Paratenic hosts of *B. procyonis* contact the eggs of the parasite in fecal material or soil associated with raccoon feces that accumulate at communal locations (Jacobson et al., 1982; Page et al., 1998). Raccoons preferentially defecate at latrines (Stains, 1956) that are predictably located at the base of large trees, or on horizontally oriented substrate including logs, tree-limbs, stumps, and rocks (Page et al., 1998). More than one raccoon may use any given latrine (Page et al., 1998), and large amounts of fecal material can accumulate (Stains, 1956). The substantial production of eggs by mature *B. procyonis* worms results in large accumulations of infective eggs at latrines (Evans, 2002b), which can remain viable for years (Kazacos, 2001). Transmission occurs when eggs are accidentally ingested during active foraging at latrines or later when animals forage on seed in fecal material that has been removed from latrines and cached (Page et al., 1999; LoGiudice, 2001). Disease can be a driver of extinction in situations where pathogens are infective to multiple hosts, and when they remain viable for prolonged periods in the environment (Smith et al., 2009; Thompson et al., 2010). *Baylisascaris procyonis* fits this description, and the latrine defecation behavior of raccoons enhances the non-specificity and the longevity of the parasite by creating a situation that increases host contact rates with the parasite.

Raccoon latrines are common throughout habitats occupied by raccoons, and in forested habitats, latrines have been reported in densities ranging from 3 to 44 latrines/ha (Page et al., 2001a; Smyser et al., 2010). Latrines also are common in urban and suburban landscapes and densities range from 8.7 to 21.7 latrines/ha (Roussere et al., 2003) or 1–6 latrines per backyard (Page et al., 2009a). The accumulation of large numbers of eggs at these latrines increases the risk to other vertebrate species that share the same habitat. *B. procyonis* eggs can persist in the environment for years (Kazacos, 2001), thus increasing the resilience of *Baylisascaris procyonis* in the environment, with eggs remaining even after the fecal material has decomposed (Kazacos, 1982; Fox et al., 1985; Cooney, 1989; Gavin et al., 2002; Kazacos et al., 2002).

## 5. Transmission dynamics of *B. procyonis* in human-dominated landscapes

Loss or conversion of habitat for human use may result in altered disease transmission dynamics (Daszak et al., 2001) by limiting the movement of potential hosts (Scott, 1988; Smith et al., 2009), and altering the contact rates of hosts and the pathogen (Smith et al., 2009). Transmission of zoonotic pathogens is influenced by the prevalence of infection in reservoir hosts, the rate at which potential hosts contact the pathogen, and the probability that infection occurs when the pathogen is contacted (Lloyd-Smith et al., 2009). Each of these factors is sensitive to landscape level modifications as host population sizes (Woodroffe, 1999; Altizer et al., 2003; Smith et al., 2009; Thompson et al., 2010) and trophic interactions (Prange et al., 2004) change with patch size and available resources.

The prevalence of *B. procyonis* seems to vary as a function of land-use (Page et al., 2001a, 2005, 2008; Kellner et al., 2012; Samson et al., 2012), and there is some information that suggests that it responds to host population density (Page et al., 2009a). While more studies are necessary to fully understand the implications of landscape heterogeneity on transmission dynamics, there does seem to be evidence that it is important. Transmission of *B. procyonis* depends on viability of eggs at latrines, thus prevalence will decrease in landscapes where environmental conditions do not allow for persistence of eggs (Kresta et al., 2010). In Texas, prevalence of *B. procyonis* was highest in ecoregions with clay soils that provided enough moisture to maintain the viability of the eggs (Kresta et al., 2010). Specific relationships of prevalence to landscape features among raccoons were higher in rural than urban portions of the Chicago metropolitan area (Page et al., 2005, 2008). Across studies, estimates for rural samples ranged from 33% to 65% and urban samples ranged from 15% to 41% (Page et al., 2005, 2008). Higher measures give a better indication of differences as they were based on visualization of worms via necropsy (Page et al., 2008) while the lower values were combined estimates from latrine, fecal samples, and necropsy analysis (Page et al., 2005). Not all studies have reported lower levels of prevalence among raccoons in urban landscapes. Blizzard et al. (2010a) reported a higher measure of *B. procyonis* among raccoons in an urban landscape (12%) than in a rural landscape in Georgia (10%), but the differences were not great. The urban areas in this study were described by the authors to be in close proximity and more similar to non-urban areas than the Chicago area studies (Blizzard et al., 2010a,b). Samson et al. (2012) conducted a survey in the Madison and Milwaukee, Wisconsin urban areas. Prevalence was not associated with urbanization, and the greatest predictors of infection were presence of agriculture and lack of forested land (Samson et al., 2012). Despite the lack of clear trends across studies, prevalence of *B. procyonis* among raccoons seems to respond to landscape-level attributes. However, it is clear that we need studies to examine the relationship between finer-scale descriptors of landscape in order to predict the level of environmental contamination (associated with latrines) and predict the risk of transmission to potential paratenic hosts.

The transmission of *B. procyonis* is dependent on the density and distribution of the raccoon definitive host; however, responses of potential paratenic hosts to landscape alterations are also important to consider. Fewer studies have examined the prevalence of *B. procyonis* infection among paratenic hosts as a function of landscape attribute; however, these studies suggest that increasing habitat heterogeneity is related to increased prevalence (Page et al., 2001a; Kellner et al., 2012). White-footed mice (*P. leucopus*) are common paratenic hosts for *B. procyonis* (Tiner, 1954; Kazacos, 2001; Page et al., 2001a). Prevalence of infection among mice has been measured in landscapes dominated by mature forest, agriculture, and urbanization (Page et al., 2001a; Kellner et al., 2012). In a study comparing prevalence across landscapes in Indiana, prevalence was higher in agriculture-dominated landscapes (28%) than in forest-dominated landscapes (6%), and across the entire landscape, prevalence increased predictably as a function of decreasing forested area and increasing isolation of patches (Page et al., 2001a). In a study of prevalence among white-footed mice in an urban landscape, overall prevalence among mice was 33%, and ranged from 17% to 42% increasing as a function of increasing human population density (Kellner et al., 2012).

If the behaviors of raccoons and the distribution of food resources are sensitive to landscape attributes, transmission of *B. procyonis* to paratenic hosts could be increased in human-dominated landscapes. In fact, changes in prevalence as a function of raccoon behavior have been reported and raccoons that were attracted to artificially aggregated food resources had increased prevalence as compared to raccoons in areas where food was more evenly distributed (Gompper and Wright, 2005). Raccoons in urban systems respond positively to artificially aggregated food sources that occur in areas of high human use (Prange et al., 2004; Bozek et al., 2007). When raccoon movements are limited to habitat patches with clumped food resources (Prange et al., 2004), and there is a disproportionate selection of habitat by raccoons (Bozek et al., 2007), the contact rate of raccoons increases, and thus could facilitate transmission of *B. procyonis*.

In landscapes where forested patches are restricted, paratenic hosts of *B. procyonis* may have increased contact rates with raccoon

latrines (Page et al., 2001a,b), thus increasing the transmission of *B. procyonis*. *Baylisascaris procyonis* transmission benefits from the attraction of a wide variety (and number) of potential paratenic hosts to raccoon latrines because large amounts of undigested seed are present in the fecal material of omnivorous raccoons (Page et al., 1999, 2001b,c; Gehrt, 2003). Numerous species of mammals (16) and birds (19) have been recorded at raccoon latrines, with active foraging also identified (Page et al., 1999, 2001b), suggesting vertebrate species that are attracted to accumulations of seed are vulnerable to transmission in habitats where raccoons are infected with *B. procyonis* and latrine densities are high. The activity and foraging behavior of a potential host while on a latrine also impacts transmission. Active foraging among fresh fecal material may not result in infection as the eggs require at least 11–14 days to become infective (Kazacos and Boyce, 1989; LoGiudice, 2001); however, active foraging for seeds embedded in old fecal material or caching of fecal material from latrines could increase transmission as eggs would have time to become infective (Page et al., 1999, 2001b,c; LoGiudice, 2001). Raccoons build latrines in predictable locations and require features associated with forested habitat patches (Page et al., 1998). Latrines may thus become concentrated in forest patches throughout a variety of landscape types, and increases in transmission to paratenic hosts increase as a function of patch size and isolation (Page et al., 2001a). Even in urban landscapes, raccoons disproportionately visit forested patches (Bozek et al., 2007), and despite decreased prevalence of *B. procyonis* among raccoons (Page et al., 2009b) in some urban landscapes, prevalence of infection among paratenic hosts in the same landscape is increased (Kellner et al., 2012). This response suggests that potential paratenic hosts may be at increased risk of infection in landscapes that alter contact rates of raccoons and ultimately latrine densities. Studies examining the density of latrines as a function of landscape attributes are needed, especially if we want to fully understand the risk of *B. procyonis* to potentially vulnerable populations of paratenic hosts.

## 6. Translocation of raccoons and other hosts spreads *B. procyonis*

Raccoons are native to North and Central America where they are widely distributed from Mexico to Canada (Lotze and Anderson, 1979; Gehrt, 2003). Introductions of raccoons have increased the range of this species within North America (Gehrt, 2003), and beyond (Beltrán-Beck et al., 2012). *Baylisascaris procyonis* ranges throughout North America, but is highest among raccoon populations in the Northeastern, Pacific, and Midwestern United States (Kazacos, 2001). The parasite also is common in Canada (Ching et al., 2000; Kazacos, 2001; Sexsmith et al., 2009) and the Appalachian Mountains (Jacobson et al., 1976; Jones and McGinnes, 1983). It is less prevalent in the Southeastern United States, but has been documented in Texas (Kerr et al., 1997; Kresta et al., 2010), Georgia (Eberhard et al., 2003; Blizzard et al., 2010a), North Carolina (Hernandez et al., 2013), Louisiana (Pai et al., 2007), and Florida (Blizzard et al., 2010b). Recent studies also demonstrate that *B. procyonis* is present in the intermountain west (Chavez et al., 2012). The extent of *B. procyonis* throughout North America suggests that the parasite can spread with raccoon populations as long as environmental conditions allow for the persistence of eggs in the environment (Kresta et al., 2010). In fact, the recent expansion of the range into Florida (Blizzard et al., 2010b) highlights the possibility of the increased threat to paratenic hosts in new ranges.

Raccoons were introduced into Europe as early as the 1920s (Frantz et al., 2005), and are currently widespread in Germany and are present in at least 20 European countries (Frantz et al., 2005; Bartoszewicz et al., 2008; Beltrán-Beck et al., 2012). The introduction of raccoons to Japan via the pet trade in the 1970s has resulted in naturalized populations in that country (Ikeda et al., 2004). The translocation of raccoons has subsequently resulted in an expansion of the range of *B. procyonis* (Miyashita, 1993; Kazacos, 2001; Bauer, 2013), which may have serious implications for small vertebrates native to these areas. When pathogens are spread via the translocation of host species, they are more likely to be linked to the extirpation of local species (Daszak et al., 2000; Smith et al., 2009; Thompson et al., 2010). While no studies have examined the prevalence of infection of *B. procyonis* among natural populations of paratenic hosts in Europe or Japan, transmission to potential paratenic hosts would be expected where prevalence is high among raccoon populations. In Germany, prevalence has been reported as high as 71% (Bauer, 2013), and there have been reported cases of baylisascariasis there and in Austria (Kazacos, 2001), suggesting that *B. procyonis* eggs are present in the environment. *Baylisascaris procyonis* has not been documented in free-ranging raccoon populations in Japan (Matoba et al., 2006); however, the parasite has been found among captive animals (Miyashita, 1993). Captive animals have been responsible for outbreaks of baylisascariasis (Kazacos et al., 1983; Sato et al., 2003), and it is possible that the parasite could escape with escaped raccoons, thus the risk to potential paratenic hosts should not be dismissed in these cases. Translocation of other infected definitive hosts, such as kinkajous (*Potos flavus*), could also spread *B. procyonis* to new areas. This was seen in the movement of the parasite into a breeding facility in southeastern Florida from kinkajous imported from the wild in Guyana (Kazacos et al., 2011). It is not known if *B. procyonis* escaped from this facility to infect local paratenic hosts or raccoons. One must also consider that translocation and release/escape of infected paratenic hosts from areas enzootic for this parasite could also spread *B. procyonis* to new areas, if they are eaten by raccoons or other potential definitive hosts.

## 7. Transmission of *B. procyonis* by domestic animals and exotic pets

The primary definitive host of *B. procyonis* is the raccoon; however, other species can harbor patent infections (Kazacos, 2001, 2006). When domestic animals are capable of transmitting a pathogen, altered transmission dynamics can increase the risk of transmission to additional hosts (Smith et al., 2009). Dogs are susceptible to patent infections with *B. procyonis* (Kazacos, 2001, 2006; Bowman et al., 2005; Lee et al., 2010), but unlike raccoons, dogs do not defecate in predictable locations. The indiscriminant defecation patterns of dogs can result in more widespread environmental contamination with *B. procyonis* eggs (Bowman et al., 2005; Lee et al., 2010). In addition to dogs, patent infections have been documented in pet kinkajous (*P. flavus*) in Tennessee and Indiana, USA (Kazacos et al., 2011). Presence of the parasite in kinkajous highlights the need to investigate the potential for other procyonids, including coatis (*Nasua* spp.), olingos (*Bassaricyon* spp.), and ringtails (*Bassariscus astutus*), to serve as definitive hosts for *B. procyonis* (Kazacos et al., 2011). It is known that *B. procyonis* ranges into the tropics where these procyonid species are common, and has been found in a kinkajou in Colombia (Overstreet, 1970). Thus, the recent expansion of *B. procyonis* to Florida (Blizzard et al., 2010b) is cause for concern. Rapid conversion of tropical habitats to human-dominated landscapes results in habitat loss and small vulnerable populations (Laurance and Useche, 2009). If *B. procyonis* is present in these ecosystems, and multiple procyonids can serve as definitive hosts, there could be serious implications for potential paratenic hosts. However, no data exist to support these assertions; therefore, this is an area in need of future study.

## 8. The role of *B. procyonis* in extinction: the Allegheny woodrat

The Allegheny woodrat (*N. magister*) is a small rodent that lives on cliff faces and talus slopes of the Eastern United States (Castleberry et al., 2006). Once distributed in these habitats throughout the Appalachian Mountain range, the species has been experiencing decline since the 1970s, and is now a species of concern through much of the range (Balcom and Yahner, 1996; Wright, 2008). Three hypotheses have been proposed to explain the decline of the Allegheny woodrat, including declines due to habitat loss, decreases in food availability, and increased mortality due to *B. procyonis* (LoGiudice, 2006). There is evidence that each of these factors has contributed in some way to the decline of the Allegheny woodrat, but the combined effect appears to be the driving mechanism (LoGiudice, 2006; Smyser et al., 2012). The conversion of hardwood forest to agriculture reduced available hard mast forage, increased the distance required to travel to find forage, and limited dispersal of individuals into portions of the range where populations had been extirpated (LoGiudice, 2006). In addition to loss of habitat, the loss of the American chestnut (*Castanea dentate*) to blight resulted in the loss of an important food source (LoGiudice, 2006; Smyser et al., 2012). Finally, *B. procyonis* has been shown to play a role in the extirpation and decline of several populations of the Allegheny woodrat (McGowan, 1993; LoGiudice, 2003; Page et al., 2012; Smyser et al., 2012).

The case of the Allegheny woodrat exemplifies how emerging pathogens, such as *B. procyonis*, can function to facilitate extinctions when factors such as habitat loss are exerting pressure on small populations. The non-specificity of *B.* procyonis with regard to paratenic hosts includes the Allegheny woodrat, which are susceptible to CNS infections with *B. procyonis* (McGowan, 1993; Kazacos, 2001; LoGiudice, 2003; Page et al., 2012). The longevity of *B. procyonis* eggs in the environment (Kazacos and Boyce, 1989) is especially problematic as woodrats frequently cache raccoon fecal material in their middens (Castleberry and Castleberry, 2008; LoGiudice, 2001). The cool, moist microclimate of caves prevents desiccation of eggs, so middens that remain after the death or dispersal of a woodrat remain a risk to any animals that subsequently take over a contaminated home range (LoGiudice, 2003; Smyser et al., 2013). It is apparent that for certain species such as the woodrat, inherent behaviors such as caching extraneous material are risk factors that contribute to transmission of *B. procyonis* with its subsequent deleterious effects on the species. The specificity of the talus slopes and cliff habitats of woodrats results in naturally limited populations that are particularly vulnerable to habitat isolation (Castleberry et al., 2006). Allegheny woodrat populations that exist in fragmented habitats often are stressed by decreased access to regular mast supply (Smyser et al., 2012). In addition, fragmented habitats decrease the ability of animals to disperse, thus limiting recruitment into small populations that could inhibit gene flow between metapopulations (Smyser et al., 2012). These small populations may be more vulnerable to infections resulting from increased contact rates with *B. procyonis* that results from the longevity of eggs at raccoon latrines and in woodrat middens. In addition to the population-level responses of woodrats to habitat fragmentation, raccoon populations and subsequently the transmission dynamics of the parasite are sensitive to landscape-level changes (Smyser et al., 2012). Raccoons and Allegheny woodrat ranges naturally overlap, so the translocation of raccoons within established areas does not play a role in the threatened status of populations. However, it is well documented in the Mohonk NY region that problems in the woodrat population did not begin until raccoons expanded their range into the area and subsequently increased in population density (Kazacos, 2001). There is no evidence that translocation of domestic animals played a role in the decline. It is clear that *B. procyonis* meets the criteria of a pathogen that can facilitate extinction, as is demonstrated by the case of the Allegheny woodrat.

Reintroductions of populations into previously occupied habitats in New York, New Jersey and Indiana highlight the challenges to conservation efforts caused by *B. procyonis* (McGowan, 1993; LoGiudice, 2003; Smyser et al., 2013). In New York, it was determined that neural larval infections with *B. procyonis* played an important role in a failed attempt to reintroduce Allegheny woodrats (McGowan, 1993). A subsequent study by LoGiudice (2003) demonstrated that the persistence of reintroduced populations of Allegheny woodrats was significantly decreased in habitats with high levels of *B. procyonis* contamination. Populations of Allegheny woodrats remain along the Ohio River in Indiana. These populations have been monitored for several decades (Cudmore, 1983; Johnson, 2002), and woodrat abundance was found to be significantly higher in habitats where *B. procyonis* was not found (Page et al., 2012). Subsequent studies of the same populations of woodrats illustrate how individual populations of Allegheny woodrats experiencing the pressures of anthropogenic land-use are more vulnerable to *B. procyonis* than populations with low levels of anthropogenic land use (Smyser et al., 2012). Finally, experimental translocations of Allegheny woodrats into previously occupied and low-density sites in Indiana included implementation of a simultaneous mitigation strategy for *B. procyonis* (Smyser et al., 2013). The successful translocations at sites receiving regularly distributed anthelmintic baits (targeting raccoons) demonstrates that consideration of *B. procyonis* in the development of conservation strategies can have significant benefits to imperiled populations (Smyser et al., 2013).

## 9. Implications of *B. procyonis* in management and conservation

Prevalence of *B. procyonis* can be measured in several different ways (Page et al., 2005). Each measure should be interpreted within the context of the life cycle, and it is important to understand how each measure is interpreted relative to transmission dynamics and assessments of risk to potential paratenic hosts. Typically, measures of prevalence are reported from studies of definitive host populations where infections were determined via fecal samples that were obtained from trapped animals or worms that were collected via necropsies (Kazacos, 2001; Page et al., 2005). If conservation decisions are made using raccoon fecal sampling, the seasonality of patent infections (Kazacos, 2001) must be considered, as estimates of prevalence using samples for spring-trapped animals may seriously underestimate patent infections. Necropsies provide researchers with more reliable estimates of prevalence among raccoon populations, as worms are visualized directly (Page et al., 2005). Larval infections among paratenic hosts can be determined, and have been used to develop transmission models (Page et al., 2001a; Kellner et al., 2012). However, determining prevalence of infection among paratenic hosts requires the killing of animals (Kazacos, 2001) and this may be counterproductive/unacceptable for species of conservation concern. However, examination of prevalence in other common paratenic hosts not of concern (such as white-footed mice) in the same localities would be an acceptable alternative and give similar information. When *B. procyonis* is a conservation concern, sampling strategies should include sampling from latrines because of their role in transmission and maintaining environmental levels of eggs (Page et al., 1999). Sampling strategies should focus on individual scats as the sampling unit, rather than the latrine (Smyser et al., 2010). Sampling single scats provides reliable estimates of transmission risk to paratenic hosts and it also provides managers with the option of sampling

a proportion of latrines that represent observed latrine size classes in the management area (Smyser et al., 2010).

Mitigation strategies could be beneficial in areas where particularly vulnerable species may be at risk for accelerated declines related to exposure to *B. procyonis*. Successful strategies to mitigate emerging zoonotic pathogens have included distributing medicated baits throughout the range of reservoir hosts (Sidwa et al., 2005; Hegglin and Deplazes, 2013). The use of anthelmintic-containing baits in conjunction with latrine removal and heat sterilization of the latrine substrate reduced environmental levels of *B. procyonis* eggs greater than 3-fold (Page et al., 2011). In addition to decreasing the environmental levels, this strategy resulted in a significant reduction in larval infections among the white-footed mouse paratenic host after one year of baiting (Page et al., 2011). Additionally, translocations of Allegheny woodrats were more successful when anthelmintic baits were regularly distributed throughout their habitat (Smyser et al., 2013). It is likely that for conservation purposes, long-term baiting strategies in habitats of high concern would be an effective strategy to lower the risk of transmission by decreasing environmental contamination with *B. procyonis* eggs, especially when combined with latrine mitigation.

## 10. Conclusions

*Baylisascaris procyonis* is a well-recognized zoonotic infection that has many of the properties associated with a pathogen capable of driving extinction. It is highly non-specific and frequently pathogenic with regard to paratenic hosts. This actually has survival value for the parasite as it facilitates transmission back to raccoons (Kazacos, 2001). With over 130 recognized species vulnerable to neural larva migrans, there are potentially many species sharing habitats with raccoons that could be vulnerable to infection. The accumulation of *B. procyonis* eggs at raccoon latrines, and the longevity of eggs in these environments allow for transmission of the parasite to vulnerable hosts for many years, even after fecal material has disintegrated. In addition, seeds present in the fecal material of omnivorous raccoons attract granivores to raccoon latrines, increasing the contact rates with the parasite and thus increasing transmission. Habitat loss, fragmentation and conversion impact the transmission dynamics of *B. procyonis*. Consistent patterns have not been elucidated; however, increases in prevalence have been recorded in association with human-dominated landscapes. In these landscapes, small vertebrates under the pressures of habitat loss or fragmentation may be more vulnerable to losses associated with *B. procyonis*. Raccoons have been introduced into new ranges, introducing *B. procyonis* to new species that may serve as paratenic hosts. The impact of *B. procyonis* on these naïve populations has not been determined, but is a potential conservation concern as exemplified by the plight of the Allegheny woodrat. In addition to transmission at raccoon latrines, environmental contamination can be exacerbated when domestic dogs maintain patent infections with *B. procyonis*. The non-specific defecation patterns of dogs could increase the contact of ground-feeding small vertebrates that may not be attracted to seeds at raccoon latrines. Other infected definitive hosts including kinkajous and coatis might play a similar role in geographic locations where they are common. Persistence of small, vulnerable populations, like the Allegheny woodrat, may be threatened when conditions arise that increase the contact of these animals with *B. procyonis*, which could have direct deleterious effects on individuals and populations. Accumulating evidence pertaining to *B. procyonis* highlights the importance of considering the role of parasitic pathogens when developing conservation strategies for vulnerable species.

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